effect (3.2, 2.5-4.2) respectively. The SI score for interaction was 3.6 (95% CI: 3.1-4.2).
Conclusions: Our study demonstrates that a familial propensity to SCA interacts with presence of increasing metabolic RF, magnifying the risks for those exposed to both.

1028-168 Is Asymmetric Dimethylarginine a Marker for Diabetes, Coronary Artery Disease, and Death/Myocardial Infarction? Results of the Intermountain Heart Collaborative Angiographic Registry Study

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Background: Asymmetric dimethylarginine (ADMA) is an endogenous inhibitor of nitric oxide synthase that has generated interest as a potential cause or marker of endothelial dysfunction and its clinical consequences, including diabetes, coronary artery disease (CAD), and renal failure.
Methods: We tested whether ADMA distinguishes patients (pt) with normal (NFG; <110 mg/dL), or impaired fasting glucose (IFG; 110-125), and diabetes (DM; ≥126 mg/dL); angiographic CAD; and death (D) or nonfatal myocardial infarction (MI) in a case-control cohort of 442 pt selected from the 3 glycemic categories from among 3000 pt entered in the Intermountain Heart Collaborative Registry. Consenting pt had fasting blood drawn for FG and ADMA during angiographic assessment and were followed for 2.6 ± 1.4 y. ADMA was assayed from cryogenically stored samples by high-pressure liquid chromatography with pre-column derivitzation and fluorescence detection. Non-parametric (KW) testing compared ADMA among groups. Logistic regression was used for predictive modeling.
Results: The study cohort consisted of equal numbers with NFG (146), IFG (148), and DM (148), matched for age (±5 y) and gender. Overall, age averaged 61 years; 72% were male; 24%, smokers; 68% had angiographic CAD; 61%, hypertension; 58%, hyperlipidemia; 24%, prior MI. During follow-up, 137 events occurred. Distribution of ADMA was broad and rightward skewed, with median 0.85 μM (range, 0-30). Mean (median) ADMA levels increased progressively by glycemic category (p<0.001): NFG=2.3 (0.74), IFG=3.0 (0.77), DM= 3.4 (1.27). When adjusted for standard risk factors, CAD severity, and presenting diagnosis, ln ADMA independently predicted DM (odds ratio [OR] 1.29 /ln unit, CI 1.07-1.54, p=0.006) and, less strongly, CAD (OR 1.21, CI 1.0-1.5, p=0.06). Ln ADMA trended higher in those with D/MI (adjusted OR 1.17, CI 0.98-1.39, p=0.08).
Discussion: Among a high coronary risk case-control cohort, ↑ADMA predicted ↑risk of IFG, DM, and CAD. Thus, ADMA might contribute to endothelial dysfunction associated with these conditions. Further research should determine whether ADMA is a causal factor or passive marker and determine causes of interpatient variability.

1028-169 The Predictive Value of Parental History of Coronary Disease

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Background: Parental history of coronary disease (CHD) is a well-known risk factor for CHD. However the risk conferred by the gender of an affected parent is controversial and data are sparse. This prospective study in high risk families was designed to determine the extent to which a maternal or paternal history of CHD contributed independently to the risk of incident CHD after adjusting for known risk factors.
Methods: Unaffected siblings (SIBS) of probands were identified from hospitalized index cases with a documented CHD event < 60 years of age. This prospective analysis includes 345 women and 339 men with either maternal, paternal or no parent with history of CHD. Parental family history was elicited by self-report and confirmed by another family member. SIBS were followed for incident CHD events. SIB events were documented by medical records and adjudicated by an external endpoints committee.
Results: The 684 SIBS were 46±7 yrs old at baseline, 50% female, 21% African American, 32% smokers, 7% diabetics, 43% hypertensives, and 67% hypercholesterolemic. Incident CHD occurred in 84/684 (12.3%) of all SIBS over 8.7±3.2 yrs follow-up. Incident events occurred in 12.9% of SIBS with no parental history, 10.5%; of sibs with a paternal history only, and 14.8% of SIBS with a maternal CHD history only. In a Cox proportional hazard analysis predicting incident CHD in SIBS, the multivariate adjusted relative risk for maternal CHD was 2.05 (95% CI=1.15-3.65) and for paternal history was 1.0 (95% CI=0.6-1.66), controlling for age, SIB sex, race, hypertension, current smoking, LDL cholesterol, diabetes, obesity and education.
Conclusion: Among individuals with a documented family history of premature CHD, having a maternal history of CHD was most strongly associated with the highest risk of a CHD event. Controlling for known risk factors, maternal history alone conferred a significant and independent excess risk of incident CHD whereas paternal alone history was not significant.

1028-170 Higher Levels of Lipoprotein-Associated Phospholipase A2 Are Associated With Higher Incidence of Cardiovascular Events at Follow-Up Independent of C-Reactive Protein

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Background: Limited data exist on the association between lipoprotein-associated phospholipase A2 (Lp-PLA2) with cardiovascular risk.
Methods and Results: We measured Lp-PLA2 levels in 504 consecutive patients undergoing clinically indicated coronary angiography. Mean age was 60±11 years and 38% were women. The mean (± SD) Lp-PLA2 level (ng/mL) was 245 ± 91. Median C-reactive protein (CRP, mg/L) was 0.29 (interquartile range: 0.12, 0.67). During a median follow-up of 4.0 years, 58 cardiovascular events occurred in 49 of 466 contacted patients (11%): cardiac death in 6, acute myocardial infarction (AMI) in 14, coronary revascularization in 28, and stroke in 10. Higher Lp-PLA2 levels were associated with a greater risk of cardiovascular events: the hazard ratio (HR) per standard deviation was 1.31 (p=0.010), and remained significant after adjusting for clinical (age, gender, smoking, hypertension) and lipid (total and HDL cholesterol, Lp(a), and triglycerides) variables and CRP (Table).
Conclusion: Higher Lp-PLA2 levels were associated with a higher incidence of cardiovascular events at follow-up, independently of traditional coronary artery disease risk factors and CRP.

Multivariate association between Lp-PLA2 and CRP with cardiovascular events at follow-up.

Endpoint	Lp-PLA2 HR (95% confidence intervals)	logCRP HR(95% confidence intervals)
Cardiac Death + AMI	1.31 (0.94, 1.81)	1.14 (0.73, 1.79)
Cardiac Death + AMI + Stroke	1.33 (1.02, 1.74)	1.26 (0.86,1.84)
Cardiac Death + AMI + Revascularization	1.25 (0.96, 1.64)	1.20 (0.88, 1.64)
Cardiac Death + AMI + Revascularization + Stroke	1.29 (1.02, 1.63)	1.26 (0.95, 1.67)
All-Cause Death + AMI	1.30 (1.01, 1.67)	1.35 (0.96, 1.89)
All-Cause Death + AMI + Stroke	1.32 (1.06, 1.65)	1.39 (1.03, 1.89)
All-Cause Death + AMI + Revascularization	1.28 (1.02, 1.61)	1.30 (0.99, 1.70)
All-Cause Death + AMI + Revascularization + Stroke	1.31 (1.06, 1.60)	1.34 (1.04, 1.72)

1028-171 Fasting Blood Glucose: An Underestimated Risk Factor for Subclinical Coronary Atherosclerosis

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Background: Non-diabetic individuals with high fasting blood glucose (FBG) are at high risk for experiencing the metabolic syndrome, which includes insulin resistance, hypertension, dyslipidemia, and a procoagulant state. While emerging evidence suggests that impaired FBG (FBG_110 mg/dl) may accelerate atherosclerosis, we sought to evaluate the independent impact of FBG in the upper normal range (<110 mg/dl) as a risk factor for subclinical coronary atherosclerosis assessed by coronary artery calcification (CAC) in an asymptomatic non-diabetic population.
Methods : We studied 531 consecutive asymptomatic, non-diabetic males (46±7 yrs; range 29-65 yrs) with FBG<110 mg/dl in the study who presented for electron-beam computed tomography (EBCT) between 1999 and 2002 in Sao Paulo, Brazil. The population was divided into 2 categories; highest quartile of FBG (>85 mg/dl, n=119) and the lowest three quartiles (n=412).
Results:Individuals in the highest quartile of FBG were more likely to have higher body mass index (28±4 vs. 26±3, p<0.0001), waist to hip ratio (0.99±0.06 vs. 0.93±0.06, p=0.01), triglycerides (410±116 vs. 180±92, p=0.05) and systolic blood pressure (131±14 vs.121±13, p<0.0001), where as no significant difference was observed in high density lipoprotein, low density lipoprotein and total cholesterol levels respectively. Overall median, 75th percentile and 90th percentile CAC scores were: 3, 24 and 156 among individuals with highest FBG quartile compared to 0, 6 and 56 among patients with lowest three quartiles (p<0.05). After adjusting for potential cofounders, the odds ratio for any calcification (CAC>0) with FBG>85 mg/dl was 2.5 (95% CI=1.1-5.4, p=0.02) and 1.8 (1.1-3.2, p=0.01) for ≥75th percentile CAC, respectively
Conclusions: FBG in the upper normal range (>85 mg/dl) appears to be an important independent predictor of presence and severity of CAC in non-diabetic apparently healthy young/middle aged men.

1028-172 Increased Urinary 8-Iso-Prostaglandin F2alpha Excretion Predict Cardiac Events in Patients With Type 2 Diabetes

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Background: Increased oxidant stress may play a key role in the etiology of diabetic cardiovascular complications. We hypothesized that increased production of 8-iso-prostaglandin F2alpha (8-iso-PGF2alpha), a marker for in vivo oxidant stress, predicts future cardiac events in type 2 diabetic patients.
Methods: We studied 148 patients aged 30-85 (62±10) with type 2 diabetes. Baseline level of urinary 8-iso-PGF2alpha excretions were measured and the patients were followed up for a mean period of 1380 days. Cardiac events were defined as hospitalization for acute myocardial infarction, unstable angina, revascularization, and worsening heart failure.
Results: One hundred thirty-nine patients completed the follow-up, while 2 died of noncardiac causes. Of the remaining 137 patients, cardiac events were occurred in 16. Urinary 8-iso-PGF2alpha excretion above the median value of 287.5 pg/mg creatinine was